Plans are already well advanced to install new generation lithotripters for treating renal stones in a number of NHS regions. Lithotripsy is currently being brought into clinical practice for the destruction of gall stones. Yet the more extravagant claims made for extracorporeal shock wave lithotripsy were not upheld in our study of routine practice. The clinical results of this first contemporaneous comparison of extracorporeal shock wave lithotripsy and percutaneous nephrolithotomy in Europe raise doubts about the superiority of extracorporeal shock wave lithotripsy over alternative techniques for treating renal calculi in two groups of patients with similar demographic and stone characteristics.

We thank Linda Jarrett, Meredith Temple-Smith, and Margot Halse, the former research assistants on the project, for their hard work in collecting much of the data, and Ann Petruckevitch for analysing the data in the late stages of preparing the manuscript. We thank the staff of the St Thomas's Lithotripter Centre and of the departments of urology at Guy's and Middlesex Hospitals, London, for access to patients' records, and Mr J Steyn and his secretary, Mrs Nealon, for providing data on patients treated at the Aberdeen Royal Infirmary. We also thank Messrs K E D Shuttleworth and M I Bultitude, consultant urologists at St Thomas's, for their advice during the design of the study and for their helpful comments on the manuscript, and two anonymous referees for constructive criticism. We gratefully acknowledge the financial support of the Department of Health and Social Security in undertaking the study.

- 1 Challah S, Mays N. The randomised controlled trial in the evaluation of new technology: a case study. Br Med J 1986;292:877-9.
  2 Palfrey ELH, Bultitude MI, Challah S, Pemberton J, Shuttleworth KED.

- Report on the first 1000 patients treated at St Thomas' Hospital by
- extracorporeal shock wave lithotripsy. Br J Urol 1986;58:573-7.

  3 Wickham J, Webb DR, Payne SR, Kellett MJ, Watkinson G, Whitfield HN. Extracorporeal shock wave lithotripsy: the first 50 patients treated in Britain. Br Med J 1985;290:188-9.
- 4 Charig CR, Webb DR, Payne SR, Wickham JEA. Comparison of treatment of renal calculi by open surgery, percutaneous nephrolithotomy, and extra corporeal shock wave lithotripsy. Br Med 7 1986;292:879-82.
- 5 Chaussy C, Schmiedt E, Jocham D, Schuller J, Brandl H, Liedl B. Extracorporeal shock-wave lithotripsy (ESWL) for treatment of urolithiasis. Urology 1984;23 (suppl):59-66
- 6 Blume E. Sound, shock waves shatter kidney stones. 7AMA 1983;249:2434-5. 7 Chaussy C, Schmiedt E. Shock wave treatment for stones in the upper urinary tract. Urol Clin North Am 1983;10:743-50.
- 8 Chaussy C, Schmiedt E. Extracorporeal shock wave lithotripsy (ESWL) for kidney stones: an alternative to surgery? Urol Radiol 1984;6:80-7.
- 9 Fuchs G, Miller K, Rassweiler J, Eisenberger F. Alternatives to open surgery for renal calculi: percutaneous nephrolithotomy and extracorporeal shock-wave lithotripsy. Klinische und Experimentelle Urologie 1984;8:153-77. 10 Miller K, Fuchs G, Rassweiler J, Eisenberger F. Financial analysis, personnel
- planning and organizational requirements for the installation of a kidney lithotripter in a urologic department. Eur Urol 1984;10:217-21.
- 11 Das G, Dick J, Bailey MJ, et al. Extracorporeal shock wave lithotripsy: first 1000 cases at the London Stone Clinic. Br Med J 1987;295:891-3.
- 12 Riehle RA, Fair WR, Vaughan ED. Extracorporeal shock-wave lithotripsy for upper urinary tract calculi: one year's experience at a single center. JAMA 1986:255:2043-8
- 13 Lingeman JE, Newman D, Mertz JHO, et al. Extracorporeal shock-w lithotripsy: the Methodist Hospital of Indiana experience. J Urol 1986;135:
- 14 Lee WJ, Smith AD, Cubelli V, Vernace FM. Percutaneous nephrolithotomy: analysis of 500 consecutive cases. *Urol Radiol* 1986;8:61-6.

  15 Sleight W, Wickham JEA. Long term follow up of 100 cases of renal calculi.
- Br J Urol 1977;49:601-4.

  16 Kaude JV, Williams CM, Millner MR, Scott KN, Finlayson B. Renal
- morphology and function immediately after extracorporeal shock-lithotripsy. American Journal of Radiology 1985;145:305-13.
- 17 Baumgartner BR, Dickey KW, Ambrose SS, Walton KN, Nelson RC, Bernardino ME. Kidney changes after extracorporeal shock-wave lithotripsy: appearance on MR imaging. Radiology 1987;163:531-4.

  18 Newman RC, Bezirdjian L, Steinbock G, Finlayson B. Complications of
- extracorporeal shock wave lithotripsy: prevention and treatment. Semin Urol 1986;4:170-4.
- 19 National Specialist Services Advisory Committee (NSSAC). Lithotripsy Health Bull (Edinb) 1987;45:2.

(Accepted 28 April 1988)

# Cigarette smoking as risk factor for late fetal and early neonatal death

Sven Cnattingius, Bengt Haglund, Olav Meirik

## **Abstract**

Risk factors for late fetal death and early neonatal mortality were examined in a population based prospective study. Practically all Swedish births between 1983 and 1985 were included, 281 808 births in all. The overall rates of late fetal death and early neonatal mortality were 3.5 and 3.1 per 1000, respectively. About 30% of the pregnant women were recorded as being daily smokers. Logistic regression analyses showed significant relative risks for late fetal death for high maternal age (1.4), nulliparity (1.4), multiparity ( $\geq$ 2) (1.3), smoking (1.4), and multiple births (2.8). Significant relative risks for early neonatal mortality were found for multiple births (4.9) and smoking (1.2). Smokers aged under 35 faced a relative risk of late fetal death ranging from 1.1 to 1.6, while the risk for late fetal death was doubled it the mothers were aged 35 years or more and smoked.

In countries like Sweden, where maternal cigarette smoking is prevalent, smoking may be the most important preventable risk factor for late fetal death.

## Introduction

The causal association between maternal cigarette smoking and low birth weight is generally accepted, whereas the association between smoking and perinatal death is disputed. In a review article McIntosh has reported that only five of 17 studies have found significantly increased risks of stillbirth among smokers.2 An increased risk of early neonatal death among the infants of smokers was reported in four studies, though none reached significance. The results of a large study of more than 360 000 single births in Missouri, United States, have also recently been published.3 In the multivariate analyses performed a significant effect of smoking on late fetal death and neonatal mortality (0-28 days) was seen. It has further been suggested that the risk of perinatal death related to smoking may not be equally distributed: it has been reported to vary with such factors as maternal age, parity, ethnic group, and socioeconomic state.4 The above studies, including the Missouri study, were based on retrospectively collected data from fairly heterogeneous populations that had perinatal mortality rates ranging from 12 to 30 per 1000.

In Sweden the overall perinatal mortality is below seven per 1000. The population is homogeneous, with poverty and undernutrition practically non-existent in the pregnant population. Antenatal and obstetric care is free, and practically all pregnant women visit the antenatal clinics regularly during pregnancy and give birth at hospitals.5 Thus Sweden provides an excellent opportunity to study late fetal death and early neonatal mortality resulting from biological variation and specific environmental influences in a well nourished population.

In 1973 a medical birth registry was set up by the National Board of Health and Welfare in Sweden.<sup>6</sup> Since 1982 information has been prospectively collected for each pregnancy from the first antenatal visit. These

Uppsala University Hospital, S-751 85 Uppsala, Sweden Sven Cnattingius, MD, associate professor in social medicine Bengt Haglund, DRMEDSC, research associate, centre for primary care research

**Human Reproduction** Programme, World Health Organisation, Geneva, **Switzerland** Olav Meirik, MD, medical

Correspondence to: Dr Cnattingius.

conditions provided an excellent opportunity to investigate the influence of smoking and other risk factors on late fetal death and early neonatal mortality in an unselected population.

#### Materials and methods

The Swedish National Board of Health and Welfare receives information on births from all hospitals about demographic data, previous reproductive history, complications during pregnancy, delivery, and the neonatal period. Since 1982 information about maternal smoking habits in the first trimester has also been included. All births and perinatal deaths reported to the birth registry are validated every year against a population registry at Statistics Sweden, which receives such information from the parishes. This cross checking is done by using the mother's personal identification number. The medical birth registry covers more than 99% of all births in Sweden.

During the study from 1983 to 1985 the number of births reported to the birth registry amounted to 282 130. Our study was restricted to births in mothers aged between 15 and 44 years. Maternal age was either unknown or outside this interval for 322 births, and these were excluded from the analyses.

Multiple logistic regression analyses were performed to estimate the effects of the studied independent variables.8 Late fetal death and early neonatal mortality were used as dependent variables. Late fetal death was defined as stillbirth occurring at 28 weeks of gestation or later and early neonatal death as death occurring during the first six completed days of life. The independent variables used in the analyses were maternal age, parity, relationship with the father, smoking habits, sex of infant, and type of birth (single or multiple). Initially all these variables were included in the analyses. In the final models, only variables that had a significant influence on late fetal death or early neonatal mortality were included. The continuous variables were categorised to avoid dubious assumptions about linearity.

Parity was defined as the number of previous live births plus stillbirths. Nulliparas were those who had

TABLE I—Number of births and deaths by maternal age, parity, relationship with father, smoking, sex of infant, and type of birth in Sweden 1983-5

		Late fetal deaths		Early neonatal deaths	
	No (%) of births	No	Death rate per 1000	No	Death rate per 1000
Maternal age at delivery (years):					
15-19	9655(3)	39	4.0	42	4.4
20-24	66 976 (24)	211	3.2	183	2.7
25-29	104 169 (37)	343	3.3	299	2.9
30-34	69 341 (25)	266	3.8	237	3.4
35-39	27 358 (10)	115	4.2	88	3.2
40-44	4 309 (2)	25	5.8	22	5.1
Parity:					
0	113 793 (40)	436	3.8	363	3.2
i	101 902 (36)	293	2.9	286	2.8
2	47 679 (17)	197	4.1	149	3 · 1
≥3	18 434 (7)	73	4.0	73	4.0
Relationship with father:	20 12 1 (17)				
Living with father	255 213 (91)	872	3.4	753	3.0
Not living with father	14 524 (5)	47	3.2	41	2.8
Information not available	12 071 (4)	80	6.6	77	6.4
Smoking habits:	12 0/1 (1/	•			
Non-smoker	182 290 (65)	563	3.1	509	2.8
Daily smoker:	102 270 (03)	202	•	507	
1-9 Cigarettes	48 834 (17)	200	4.1	169	3.5
≥10 Cigarettes	31 458 (11)	141	4.5	92	2.9
Information not available	19 226 (7)	95	4.9	101	5.3
Sex of infant:	17220(1)	,,	• /	•••	
Boy	144 976 (51)	526	3.6	469	3.2
Girl	136 249 (48)	469	3.4	389	2.9
Information not available	583 (0)	107	٠,	307	
Type of birth:	202 (0)				
Single	276 269 (98)	950	3.4	793	2.9
Multiple	5 534 (2)	49	8.9	78	14.2
Information not available	5(0)	42	3,7	76	14.2
Total	281 808 (100)	999	3.5	871	3·1

had no previous births. Information about relationship with the father and smoking habits was obtained by midwives at the women's first antenatal visit. Nowadays it is common in Sweden for couples to live together without being married. Information about relationship with the father was therefore recorded as the mother living with the infant's father or not. Smoking habits were classified as non-smoker (that is, non-daily smoker), smoking between one and nine cigarettes a day, and smoking 10 cigarettes or more a day. In view of the increasing knowledge among mothers about the effects of smoking on the outcome of pregnancy the daily number of cigarettes smoked may have been underreported. Thus such a quantitative assessment of smoking habits may be considered to be rather uncertain. In the logistic regression analyses performed smoking habits were therefore simply classed as daily smoker or not.

The statistical analyses were performed with the SAS program package. 9 10

### Results

Table I shows the number of births and mortality according to the independent variables included in the analyses. The table indicates that teenage pregnancies, high maternal age, nulliparity, high parity ( $\geq 2$ ), multiple births, and smoking were associated with increased numbers of late fetal deaths. There also seemed to be a dose-response relation between the number of cigarettes smoked a day and late fetal death rate. Information about maternal smoking habits and relationship with the father was not available for 19226 (7%) and  $12\,071$  (4%), respectively, of the mothers. The small number for whom information on smoking and relationship with the father was missing had quite high rates of fetal and early neonatal mortality (table I). The risk of early neonatal death was particularly associated with multiple births. Neither late fetal nor early neonatal mortality was higher among women who reported not living with infant's father.

About 30% of the studied population were daily smokers, but smoking habits varied among different groups in the population (table II). Half of the teenage mothers smoked compared with 25-29% of mothers aged 25 or more. Of women who were not living with the infant's father, 54% were daily smokers, whereas 29% of women who were living with the infant's father smoked.

These variations in smoking habits required a multivariate approach. Table III shows the influence of age, parity, smoking, and type of birth on late fetal death. The baseline risk represents an estimate of the risk of late fetal death among low risk women (that is, non-smoking women aged 20-29 with one previous birth and the present birth being a single birth). As can be seen from table III, smoking increased the predicted late fetal mortality from 2.2 to 3.0 per 1000 (relative risk=1.4). Other factors that significantly influenced the rate of late fetal death were high maternal age, nulliparity, high parity ( $\geq 2$ ), and multiple births. In table III only births that had available information on the independent variables were included. Assuming that all women with missing data on smoking habits were non-smokers or smokers changed the relative risks for smoking only marginally (relative risks=1.3 and 1.4, respectively).

Table IV shows the rates and relative risks of early neonatal mortality according to the same factors as in table III. Compared with that in late fetal death, the influence of smoking on neonatal mortality was lower and of marginal significance. High maternal age, nulliparity, and high parity were not significantly related to neonatal mortality. For multiple births the risk of early neonatal mortality was higher than the

			No (%) of daily smokers		
	No	No (%) of non-smokers	<10 Cigarettes/day	≥10 Cigarettes/day	
Maternal age at delivery (years	s):				
15-19	8 871	4 427 (50)	2 770 (31)	1 674 (19)	
20-24	62 597	38 814 (62)	14 588 (23)	9 195 (15)	
25-29	97 458	69 572 (71)	17 221 (18)	10 665 (11)	
30-34	64 530	47 751 (74)	10 169 (16)	6 610 (10)	
35-39	25 218	18 791 (75)	3 575 (14)	2 852 (11)	
40-44	3 908	2 935 (75)	511 (13)	462 (12)	
Parity:					
0	106 137	72 296 (68)	21 413 (20)	12 428 (12)	
ì	95 081	67 648 (71)	16 851 (18)	10 582 (11)	
2	44 394	30 979 (70)	7 718 (17)	5 697 (13)	
≥3	16 970	11 367 (67)	2 852 (17)	2 751 (16)	
Relationship with father:					
Living with father	244 389	173 005 (71)	44 259 (18)	27 125 (11)	
Not living with father	13 498	6 233 (46)	3 583 (27)	3 682 (27)	
Information not available	4 695				
Total	262 582*	182 290 (69)	48 834 (19)	31 458 (12)	

<sup>\*</sup>Information on maternal smoking habits was not available for 19226 deliveries.

TABLE III—Predicted rates and relative risks of late fetal births based on a logistic regression model, Sweden 1983-5 (total 262 577 births)

	Predicted death rate per 1000	Relative risk (95% confidence interval)
Maternal age (years):		
15-19	2.2	1·0 (0·7 to 1·4)
20-29 (reference group)	2.2	1.0
30-34	2.8	1·3 (1·1 to 1·5)
35-44	3.0	1·4 (1·1 to 1·7)
Parity:		
0	3.1	1.4 (1.2 to 1.7)
1 (reference group)	2.2	1.0
≥2	2.8	1·3 (1·1 to 1·5)
Smoking habits:		
Non-smoker (reference group)	2.2	1.0
Smoker	3.0	1·4 (1·2 to 1·6)
Type of birth:		
Single (reference group)	2.2	1.0
Multiple	6.1	2.8 (2.1 to 3.8)

TABLE IV—Predicted rates and relative risks of early neonatal death based on a logistic regression model, Sweden 1983-5 (total 261673 births)

	Predicted death rate per 1000	Relative risk (95% confidence interva
Maternal age (years):		
15-19	2.8	1·3 (0·9 to 1·8)
20-29 (reference group)	2.2	1.0
30-34	2.8	1.2 (1.0 to 1.5)
35-44	2.7	1·2 (0·9 to 1·5)
Parity:		
0	2.6	1·1 (0·9 to 1·4)
1 (reference group)	2.2	1.0
≥2	2.4	1·1 (0·9 to 1·3)
Smoking habits:		
Non-smoker (reference group)	2.2	1.0
Smoker	2.6	1·2 (1·0 to 1·4)
Type of birth:		
Single (reference group)	2.2	1.0
Multiple	11.0	4.9 (3.8 to 6.4)

risk of late fetal death (relative risks=4.9 and 2.8, respectively).

From the viewpoint of primary prevention smoking was the only risk factor included that could, at least theoretically, be avoided. It was therefore interesting to calculate the risk attributable to smoking for late fetal death and early neonatal mortality. These risk measures were used to estimate the possible reduction in mortality if smoking were eliminated and the infants of mothers who gave up smoking faced the same mortality risks as their non-smoking counterparts (that is, same maternal age, parity, type of births, and so on). We estimated that late fetal death rate would be reduced by 11% and early neonatal mortality by 5% if smoking could be eliminated from the pregnant population (in all about 50 deaths a year in Sweden).

To study whether smoking increased the risk of late fetal death or early neonatal mortality more for certain women than for others interactions between smoking and the other variables were analysed. For late fetal death the analyses indicated an increased risk with increasing maternal age and parity, though this risk increase was not significant (p=0.20). A significant interaction between smoking and high maternal age was, however, obtained for late fetal death (p<0.05). No such synergistic effect of smoking and maternal age (or other risk factors) could be seen for early neonatal mortality.

To specify further the possible but not clear interaction between smoking and high maternal age for late fetal death separate logistic regression analyses were performed for the different age groups. As table V shows, the relative risk of late fetal death for smokers v non-smokers ranged from 1·1 to 1·6 in the age groups below 35, while the relative risk was 2·0 among women aged 35 to 44. In view of the variations in smoking

TABLE V—Adjusted relative and attributable risks for late fetal death among smokers by maternal age in Sweden 1983-5, based on logistic regression models

Age group (years)	Relative risk (95% confidence interval)	Attributable risk (%)
15-19	1·1 (0·5 to 2·2)	3
20-24	1.6 (1.2 to 2.1)	17
25-29	1·3 (1·0 to 1·6)	7
30-34	1·3 (0·9 to 1·6)	6
35-44	2·0 (1·4 to 2·9)	21
15-44	1·4 (1·2 to 1·6)	11

habits in the different age groups (table II) and the possible different effects of smoking in these groups the attributable risks of smoking were calculated for each age group. The attributable risk of smoking for late fetal death was not equally distributed in the different age groups: it ranged from 3% to 21%. Among women aged 20 to 24 the attributable risk of smoking was estimated to be 17%; 38% of these women were reported to be daily smokers (table II). Among women aged between 35 and 44 years 21% of late fetal deaths were attributable to smoking, though only a quarter of these women smoked.

## Discussion

Though the concept of perinatal mortality is useful in clinical practice, it may not be as useful in analytical studies. Because some risk factors are primarily associated with late fetal death and others more with early neonatal mortality the importance of a specific risk factor might be concealed with the broader concept of perinatal mortality as the endpoint variable. Retarded fetal growth is generally considered to be the main risk factor for late fetal death, while for early neonatal mortality preterm birth has been put forward as the most important risk factor.11 The association between smoking and retarded fetal growth is stronger than that between smoking and preterm birth. 12 Thus it seems logical that the increased rate of perinatal death observed among infants of smokers may primarily be due to an increased risk of late fetal death.

It has previously been suggested that smoking does not increase the risk of perinatal death among women who in other respects are at low risk.<sup>13</sup> The present results indicate that smoking is one of the main risk factors for late fetal death even in a low risk population. We observed increased risks of late fetal death among older women, and this risk doubled if the women smoked. This is in accordance with the results of Meyer *et al*<sup>14</sup> and also agrees very well with the previously reported interaction between smoking and

maternal age with regard to retarded fetal growth, 18 which in turn is associated with fetal death.

Teenage pregnancies have been reported to be associated with a slightly increased risk of perinatal mortality. In our study high crude rates of late fetal death and neonatal mortality were seen among the infants of teenage mothers (table I). In the multivariate analyses performed low maternal age disappeared as a risk factor, as these mothers in our population were often nulliparas, and half were smokers. Thus our data suggest that the higher mortality seen for infants of women in their upper teens are caused by environmental influences rather than a specific negative effect of low maternal age.

We observed a strong association between smoking and late fetal death when controlling for effects of factors like maternal age, parity, relationship with father, and type of birth. Other unmeasured factors are unlikely to influence appreciably the magnitude of the association found between smoking and late fetal death.3 Relationship with the father was the only socioeconomic variable available in this study; in other countries it has previously been reported to be appreciably associated with late fetal death.317 In our study, however, no associations were found between the relationship with the father and late fetal death or early neonatal mortality. Furthermore, it is generally agreed that socioeconomic differences in Sweden are small, and a previous Swedish study failed to obtain significant differences in perinatal mortality with regard to different socioeconomic variables.7 The present study was a prospective population based study that included more than 280 000 births. The results give strong support to the hypothesis of a causal relation between smoking and late fetal death.

Our present information on smoking habits was based on the mothers' reports to the midwives during their first visit for antenatal care. Today pregnant women are subjected to massive antismoking publicity and a generally increasing intolerance towards smoking in society. This may have influenced the validity of the self reported statements about smoking habits, causing the number of smokers to be underestimated. Smokers may also have stopped smoking after their first visit for antenatal care. It has been shown in a randomised controlled trial that the infants of mothers who stopped smoking in early pregnancy gain more weight during pregnancy than those of mothers who continued to smoke during pregnancy.18 The real association between smoking and late fetal death may therefore be greater than that seen in this study.

During recent decades perinatal mortality has con-

tinued to decrease dramatically in industrialised countries. Smoking is at present probably the most important preventable risk factor for late fetal death. In the early 1960s, when the dangers of smoking during pregnancy were not known to the public, 97% of Swedish smokers continued to smoke during pregnancy. Today pregnancy is probably a unique incentive for women to stop smoking. As many as 20% stop smoking "on their own" shortly after becoming pregnant, and adequate support may motivate another 20% to give up smoking. Though the main reason to stop smoking during pregnancy has primarily been put forward as concern for fetal wellbeing, the potential long term effects of improved maternal health may prove to be of equal importance in the future.

We are indebted to Anders Ericsson and Jan Gunnarskog at the National Board of Health and Welfare for providing data for this investigation.

- Bakketeig LS, Hoffman HJ, Oakley AR. Perinatal mortality. In: Bracken MB, ed. Perinatal epidemiology. Oxford: Oxford University Press, 1984:99-151.
- 2 McIntosh ID. Smoking and pregnancy. II. Offspring risks. Public Health Rev 1984;12:29-63.
- Kleinman JC, Pierre MB, Madans JH, Land GH, Schramm WF. The effects of maternal smoking on fetal and infant mortality. Am J Epidemiol 1988;127:274-82.
- Meyer MB, Jonas BS, Tonascia JA. Perinatal events associated with maternal smoking during pregnancy. Am J Epidemiol 1976;103:464-76.
   Rooth G. Better perinatal health. Lancet 1979;ii:1170-2.
- 6 Karlberg P. Medical birth registration. In: Chalmers I, McIlwaine G, eds. Perinatal audit and surveillance. London: Royal College of Obstetricians and Compressionists. 1980:221-7
- Gynaecologists, 1980:221-7.
  7 Ericsson A, Eriksson M, Westerholm P, Zetterström R. Pregnancy outcome and social indicators in Sweden. Acta Paediatr Scand 1984;73:69-74.
- and social indicators in Sweden. Acta Paediatr Scand 1984;73:69-74.
  8 Schlesselman J. Case-control studies. Design, conduct, analyses. New York: Oxford University Press. 1982:354.
- 9 SAS Institute. SAS user's guide: basics. Version 5. Cary, North Carolina: SAS Institute, 1985:1290.
- 10 SAS Institute. SAS user's guide: statistics. Version 5. Cary, North Carolina: SAS Institute, 1985;956.
- 11 McIlwaine GM, Howat RCL, Dunn F, Macnaughton MC. The Scottish perinatal mortality survey. Br Med J 1979;ii:1103-6.
- 12 Fredrick J, Adelstein P. Factors associated with low birth weights of infants delivered at term. Br 7 Obstet Gynaecol 1978;85:1-7.
- 13 Werler MM, Pober BR, Holmes LB. Smoking and pregnancy. Teratology 1985;32:473-81.
- 14 Meyer MB, Tonascia JA, Buck C. The interrelationship of maternal smoking and increased perinatal mortality with other risk factors. Further analyses of the Ontario perinatal mortality study, 1960-61. Am J Epidemiol 1975;100: 443-52
- 15 Cnattingius S, Axelsson O, Eklund G, Lindmark G. Smoking, maternal age and fetal growth. Obstet Gynecol 1985;66:449-52.
- 16 McCormick MC, Shapiro S, Starfield B. High risk young mothers: infant mortality and morbidity in four areas in the United States, 1973-1978. Am J Public Health 1984:74:18-23.
- 17 Butler NR, Bonham DG. The first report of the British perinatal mortality survey. Edinburgh: E & S Livingstone, 1963:14-26.
- Edinburgh: E & S Livingstone, 1963:14-26.

  18 Sexton M, Hebel RH. A clinical trial of change in maternal smoking and its effects on hirthweight. TAMA 1984:251-2911-5.
- effects on birthweight. JAMA 1984;251:911-5.
   19 Kullander S, Källen B. A prospective study of smoking and pregnancy. Acta Obstet Gynecol Scand 1971;50:83-94.
- 20 Kruse J, Le Fevre M, Zweig S. Changes in smoking and alcohol consumption during pregnancy: a population-based study in a rural area. Obstet Gynecol 1986;67:627-32.

(Accepted 10 May 1988)

## ONE HUNDRED YEARS AGO

THE prescription "a voyage in a sailing ship" is sometimes too lightly given to patients suffering from pulmonary disor 'er. Some of the discomforts as well as the actual dangers to such patients, which must be run, are well described in a letter recently addressed to his physician by a medical man in delicate health who sailed for New Zealand last autumn; he says:—

"I think it would be a matter of much importance to point out to medical men how much depends on the ship in sending patients for a long sea voyage. Many of them—London medical men—are quite unaware of the risk and actual hardships that have to be undergone on board sailing ships not purposely fitted up for the comfort of passengers. Most people now travel by steamer, and so but little care is taken regarding the victualling and furnishing of the sailing ships. The hospital ships as they are called—the Sobraon, for instance, of the Devitt and Moore line—are, of course, exceptions, and by them invalids should go, or perhaps by the other ships specially fitted for the passenger trade. Besides not carrying food of quality and variety suited to invalids, ordinary ships go much too far south for chest cases. We were for about five weeks between latitude 45° and 47° S.,

and though it was midsummer the cold was intense, and the heavy sea and wet decks obliged us to keep below for a great part of this time. On one occasion a heavy sea broke over the ship and flooded the saloon and cabin, everything was wet, and for a week or more the place was very damp, and the stove-heat caused constant steam. You may imagine how the invalids suffered. I mention these particulars to enable you to form a judgment of what happens in the usual southern course of the sailing ships. The ships I mentioned above, Sobraon, etc., do not go south beyond the Cape, after passing which they at once steer north again, and go to the east in latitude 30° to 40°, so that they never have cold, bleak weather the whole way to Australia. In fact, my experience amounts to this: that invalids lose as much during the latter part of the voyage to New Zealand as they gain by the early part (in the ordinary ships). I am sure you will believe me when I say this is a strictly accurate account. I am not actuated by any feeling of disappointment in my own case, but merely by a desire to warn other invalids of the risks so little known.'

(British Medical Journal 1888;i:811)